A novel lesion of the infraspinatus characterized by musculotendinous disruption, edema, and late fatty infiltration

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Isolated, atraumatic rupture of the infraspinatus (IS) with associated edema of its muscle is a previously undescribed lesion. We retrospectively identified 19 patients with MRI detected, isolated lesions of the IS tendon or musculotendinous junction with associated muscle edema. The average age at the time of presentation was 47.7 years (range, 30-66). There were 15 females and 4 males. Fourteen patients were treated nonoperatively; 5 underwent an open repair. All patients underwent clinical and MRI follow-up at an average of 50.3 months (range, 24-79) after the initial MRI. Constant scores were recorded as was a detailed physical examination. Two patients had a clear history of trauma with no preceeding shoulder problems; 17 had a history of chronic shoulder pain. We identified disruption occurring within the IS tendon in 9 patients and at the level of the musculotendinous junction in 8, while the level of disruption was inconclusive in 2. No patient had other full thickness tears. Electromyographic studies were normal, as were nerve conduction velocities in the suprascapular nerve in the 15 patients tested. There was a significant improvement in the Constant score comparing the score at presentation (53 points; range, 24-69) with the score at final follow-up (67.2 points; range, 28-95; P *= .009). There was no significant benefit seen comparing the gain in the Constant score between those treated operatively and nonoperatively (*P *= .61). All 19 patients had progressed to stage 4 fatty infiltration of the IS muscle at latest follow-up. (J Shoulder Elbow Surg 2008;17:546-553.)*

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Edema of the infraspinatus is a rare entity. It has been described in cases of denervation, such as in Parsonage Turner syndrome[3,16] or with compression of the suprascapular nerve.[20] The latter is most commonly due to a paralabral ganglion but can also be due to tumors and vascular malformations adjacent to the nerve in the spinoglenoid notch.[6,14] In all of these causes, the structural integrity of the infraspinatus is intact.

Muscular edema is characterized on magnetic resonance imaging (MRI) by high signal intensity on T2-weighted images and represents increased intra- or extracellular water content. This ingress of water may be traumatic,[13] neurogenic,[12] or inflamatory.[21,29] Other conditions, such as sickle cell disease,[11] infection,[33] early stage myositis ossificans,[19] Graves disease,[5] and mass lesions, such as tumors,[22] may also result in localized tissue edema.

Complete tendon rupture has been associated with muscular edema, although this is not a universal finding. Rupture of the distal biceps tendon and the conjoint tendon of the hamstrings both may cause muscular edema.[4,10] Injury to the musculotendinous junction in muscles, such as the pectoralis major,[7,26] biceps brachii,[1] hip adductors,[8] and others,[28] has been classified into 3 stages:[37] muscle stretch injuries (type 1), which heal without adverse sequelae; partial rupture (type 2), in which there is no tendon retraction; and complete rupture (type 3), where the muscle belly retracts. High signal on T2 weighted images are typical of all 3 stages. This high signal is seen in the gap in cases of complete rupture, tracking along muscle fasicles. This response has been well-demonstrated.

The MRI diagnosis of an isolated lesion of the musculotendinous junction of the infraspinatus in association with muscular edema was recently described.[32] Our aim with this study was: (1) to identify, illustrate, and understand this pathology affecting the infraspinatus muscle, and (2) to establish its natural history and try to clarify the treatment.

MATERIALS AND METHODS

We prospectively identified 19 patients (Table I) with isolated infraspinatus rupture and a characteristic edema pattern of the infraspinatus muscle on MRI over a 6-year

Table I Patient demographic, radiographic, MRI and outcome data

ID	Age tendon	Dom	Sex	Work arm	Ca++	Traumatic	Surgery onset	MRI Infra remnant
1	59	Y	F	Doctor	Y*	N	Y	Y
2	37	Y	F	At home	Y	N	N	N
3	60	Y	M	Radiographer	N	N	N	N
4	53	Y	F	Doctor	Y	N	N	Y
5	51	Y	F	Not working	Y*	N	N	Y
6	38	Y	M	Locksmith	N	N	Y	N
7	53	Y	F	Technician	N	N	N	Y
8	44	N	F	Teacher	Y*	N	N	N
9	50	N	F	Not working	N	N	N	Unk
10	52	N	F	Secretary	Y	N	N	N
11	30	N	F	Seamstress	N	N	N	N
12	42	N	M	Shop keeper	Y	N	Y	N
13	44	Y	F	At home	Y	N	N	N
14	66	N	F	Secretary	Y	N	N	Y
15	41	Y	F	Not working	Y	N	N	Y
16	47	N	F	Life guard	Y*	N	Y	N
17	45	Y	M	Salesperson	Y	Y	Y	Y
18	30	N	F	Bar staff	N	Y	N	Unk
19	49	Y	F	Secretary	N	N	N	Y

ID	Duration of follow-up†	Constant score normal side	Constant score presentation	Constant score review	Workers compensation
1	63	91	68	49	Y
2	25	89	55.5	77.5	N
3	50	89	51	82.5	N
4	33	81	50	37.5	Y
5	46	91	53	51.5	Y
6	54	91	62	78	N
7	33	85	32	78.5	N
8	60	89	24.5	75	N
9	24	87	27.5	28	N
10	72	89	54	74	Y
11	50	87	42.5	62.5	N
12	55	95	32	85.5	N
13	38	89	69	76	N
14	74	87	60.5	87	N
15	25	85	64.5	71.5	N
16	49	85	53	44	Y
17	79	95	78.5	95	N
18	66	89	65	61	Y
19	60	89	65	78.5	N

Dom, Dominant.
*Aspiration of calcific deposit.
†months.

period from 1998 to 2004. The individuals were identified by typical findings from all the patients with MRI scans for shoulder pain. All patients had no other full thickness tears within the rotator cuff. None had bilateral disease. The average age was 47.7 years (range, 30-66.5). There were 15 females and 4 males. The dominant extremity was involved in 12 and the nondominant in 7. Eight patients participated in regular exercise, with only 1 engaging in overhead sports (#17, Table I).

All patients underwent clinical examination at the time of initial presentation. Needle electromyographic studies of the rotator cuff muscles, looking individually at the supra and infraspinatus, and nerve conduction studies of the upper limb, looking at motor and sensory conduction velocities, were carried out on 15 of 19 patients. The mean time from onset of acute symptoms to neurophysiological assessment was 10.5 months (range, 5-22). All patients were reexamined at follow-up and Constant scores assessed.

All patients had anteroposterior radiographs in 3 different rotations (internal, external, and neutral) and supraspinatus outlet views, as well as MRI scans with both T1 and T2 weighted images at presentation and at follow-up. All MRIs were analyzed for the presence of mass lesions (in particular at the spinoglenoid and suprascapular notches), the

appearance of the tendon and musculotendinous junction, the presence or absence of edema seen on T2 images, and fatty infiltration seen on T1 images. Fatty infiltration, if present, was graded.[15] Edema of the infraspinatus was best seen on T2, or T2 fat saturated, weighted images. The tendon, aponeurosis, and any scar tissue are dark on both T1 and T2 weighted images. In the normal infraspinatus, sagital images clearly show the normal tendon and its transition into the curvilinear aponeurosis. The aponeurosis can be followed medially, where it becomes thinner and moves to the posterior margin of the infraspinatus muscle.

Patients were treated either nonoperatively (14 patients) with physiotherapy conducted in a pool, activity modification, and the judicious use of steroid injections under fluoroscopic control or surgically (5 patients). Surgical intervention was performed through an open lateral approach; 3 had a lesion of the infraspinatus tendon characterized by discontinuity and severely disorganized tissue A direct repair was performed by muscle to tendon or tendon to bone sutures with nonabsorbable material.

Statistical analysis was carried out using GraphPad Prism software version 3.00 (San Diego, CA). Data was analyzed using the Mann-Whitney test for nonparametric data, Chi-squared analysis for 2 x 2 tables, and unpaired *t*-tests for parametric data. The level of significance was set at $P < .05$.

RESULTS

Patients had a varied onset of symptoms. Two had a clear history of trauma (patients 17 and 18, Table I) with no preceding shoulder pain. Seventeen had a history of chronic shoulder pain, averaging 4.3 years (range, 6 months-10 years). In 15, an episode precipitating a different and more severe pain was superimposed, leading to their index MRI scan.

The primary complaint was pain, with all having night pain. This pain was constant, but eased by limiting the use of the arm and by keeping the elbow close to the side. Activities, such as lifting a heavy object, changing gears in a car, or using the arm overhead, were associated with severe pain. This acute initial stage lasted between 6 weeks and 1 year, with an average duration of 2.5 months. The average absolute Constant score at presentation was 53 points (range, 24.5-69), compared to an average score of 89 points (range, 81-95) on the unaffected side (Table II).

At presentation, strength in external rotation with the elbow at side was decreased in all individuals; 13 had MRC grade 4 /5 power and 6 had MRC grade 3/5 power in external rotation. No patient had a positive Hornblowers sign, and the strength in external rotation at 90° of abduction was normal (MRC grade 5/5). Clinical neurological examination was normal in all patients. Electromyographic studies on the deltoid and rotator cuff were normal, as were nerve conduction velocities in the suprascapular, median, ulnar, and radial nerves in the 15 patients tested.

Overall, only 1 patient did not have a shoulder injection performed by their general practitioner prior to the index MRI, with the average number being 2.3 (range, 0-5) . Shoulder and/or bursal injections in 2 cases and needle aspiration of calcific tendinitis in 3 were attributed directly by the patients to the acute deterioration in their symptoms and with their onset of weakness.

Table II Mean Constant scores at onset and at review. Each category of the Constant score compares patients who were operated on and those treated nonoperatively

	Mean score at presentation		Mean score at review	
Category*	Non-op†	Op†	Non-op†	Op†
Pain	4.9	5.1	9.5	9.3
Function	9.7	10	14.8	15
Movement	30.3	32.8	34.8	34
Power	8.1	10.8	7.5	11.2
Total (% opposite side)	51(58.3%)	53.0(58%)	66.6(75%)	69.5(75.4%)

$P = .009$ when comparing total constant scores at presentation with those at follow-up.
$P = .61$ when comparing change in total constant scores between the operated and nonoperated groups.
*Maximum scores for each category are pain =15, Function 20, Movement 40, power kg × 2 held in 90° flexion.
†*Non-op Op*, Non-operated Operated .

Plain radiographs showed no evidence of arthritis or narrowing of the acromio-humeral distance. Calcific deposits at the rotator cuff insertion were seen in 13 individuals. Calcification was isolated to the supraspinatus insertion in 7 cases, to the infraspinatus in 2, and to both tendons in 4. Calcification was homogeneous in 5 cases, homogeneous and multilobulated in 5, and heterogenous in 3.

MRI scanning showed no evidence of a mass lesion either in the suprascapular notch or at the base of the spinous process. Two characteristic features were seen in the infraspinatus muscle on MRI; these were tendinous or musculotendinous rupture and edema. We identified disruption on the sagittal view (Figure 1), as occurring at the level of the tendon in 9 patients and at the level of the musculotendinous junction in 8. In 2 patients, identification of the exact level was inconclusive. More medially in the muscle belly, when the aponeurosis reappears, it was thickened with loss of its normal contour, suggesting retraction after rupture. This change in the aponeurosis was best appreciated on the axial views (Figure 2). The coronal oblique view also showed an apparent discontinuity (Figure 3), although the oblique course of the infraspinatus implies that it is not all in the same plane on any one image.

Edema appeared as high signal seen on either T2 or T2 fat saturated images. These showed features typical of a tendinous or musculotendinous injury, with fluid at the junction tracking around the muscle

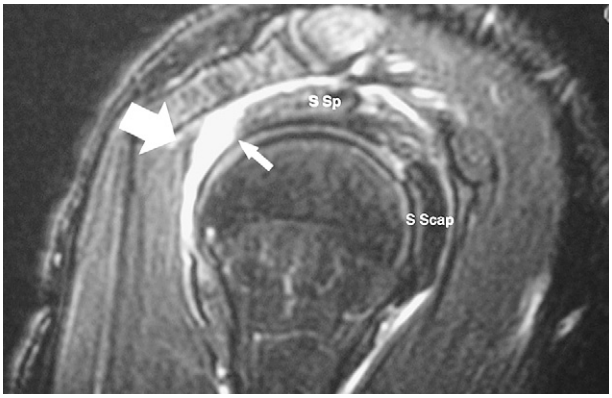

Figure 1 Sagittal T2 weighted MRI scan showing absence of the infraspinatus muscle (*large white arrow*) and articular cartilage (*small white arrow*). (Patient 10, Table I): *S Sp*, Supraspinatus; *S Scap*, Subscapularis.

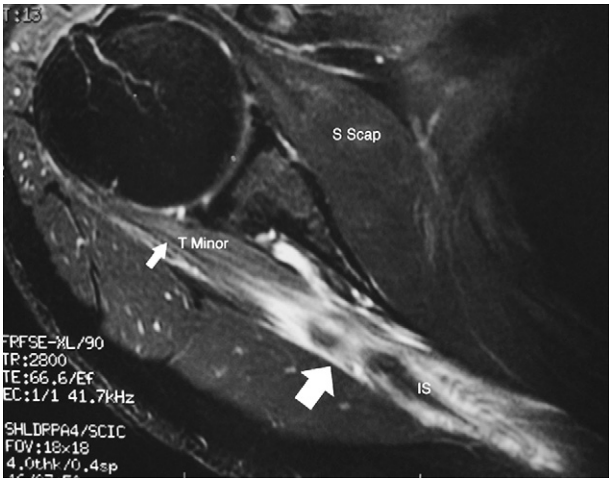

Figure 2 Axial T2 weighted MRI scan showing edema in the infraspinatus muscle in association with retraction and thickening of the aponeurosis (*white arrow*). (Patient 19, Table I): *T minor*, Teres Minor; *S Scap*, Subscapularis.

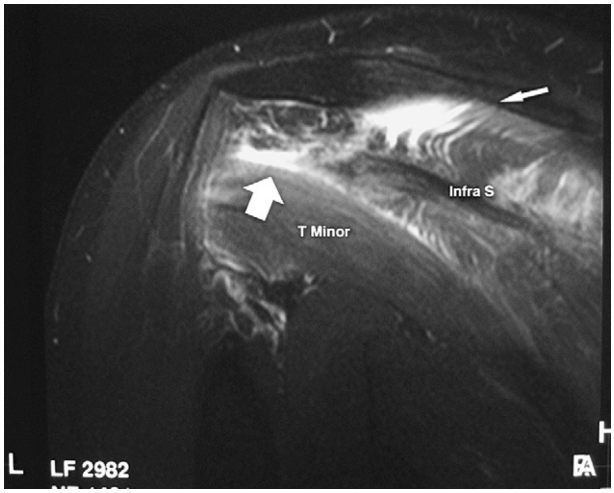

Figure 3 Coronal T2 weighted MRI scan showing high signal at the site of the disruption (*large white arrow*) and an increased pennate angle (*small white arrow*) due to muscular retraction of the infraspinatus. Edema is also seen in the infraspinatus muscle belly. (Patient 1, Table I): *T Minor*, Teres Minor; *Infra S*, Infraspinatus.

fascicles and involving the entire infraspinatus muscle (Figure 3). This edema was well seen on all 3 views (Figures 4-6).

Seven patients had CT arthrograms. In 6 of these patients, no leak of contrast was seen from the glenohumeral joint. One, who had a traumatic onset, had a contrast leak into the musculotendinous junction of infraspinatus.

All patients underwent clinical and MRI follow-up. The average duration of clinical follow-up was 50 months (range, 24-79). Constant scores were calculated. There was a significant improvement in the total score, when comparing the score at presentation with the score at final review ($P = .009$) (Table II). There was no significant benefit seen when comparing the gain in Constant score between those who were operated on and those treated nonoperatively ($P = .61$) (Table II). Six patients were involved in Workers' Compensation and had an average Constant score of 52.8 (range, 37.5-74). The remaining 13 had an average score of 77.5 (range, 28-95). The 6 patients involved in Workers' Compensation were the only ones to have a worse Constant score at review, falling an average of 10.6 points, whereas the remaining 13 improved their Constant score on average of 23 points (range, 1-50.5).

All patients had pronounced wasting of the infraspinatus muscle with a resulting concavity of the infraspinous fossa. Range of motion testing showed some limitation compared to the normal side. Average active elevation was 158°/176° (affected/normal sides), average external rotation of 48°/58°, and internal rotation placing the hand on the back limited, with the affected side having a median level of thoracic vertebra 12 and the unaffected of thoracic vertebra 7. No patient had an abnormality on testing of the subscapularis. No patient had a positive Hornblower's sign.

Strength testing showed power in external rotation with the elbow at side to be significantly less than the normal side ($P < .0001$), with a mean of 3.2 kg on the affected side and a mean of 6 kg on the normal side.

At the time of their most recent MRI at a mean of 42 months (range, 24.7-80.1 months) after the index MRI, all patients had stage 4 fatty infiltration of the infraspinatus muscle. Fatty infiltration, seen on T1 images, was best appreciated on the axial view, although it was also well seen on coronal and sagittal views (Figures 7 and 8). Associated MRI detected rotator cuff pathology were found in 11 patients and

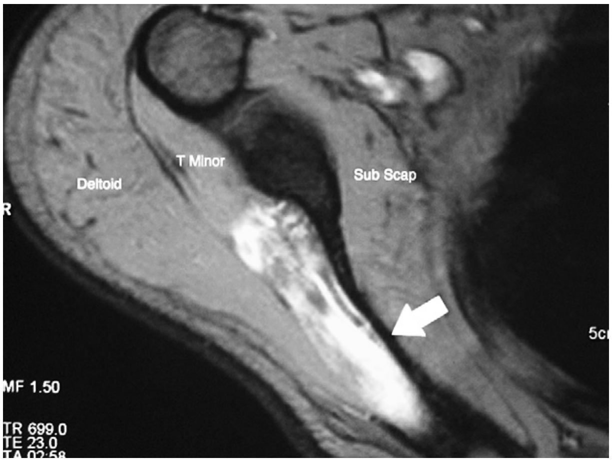

Figure 4 Axial fat saturated T1 weighted MRI with intravenous gadolinium contrast showing high signal in the infraspinatus muscle (*white arrow*). (Patient 2, Table I): *T Minor*, Teres Minor; *S Scap*, Subscapularis.

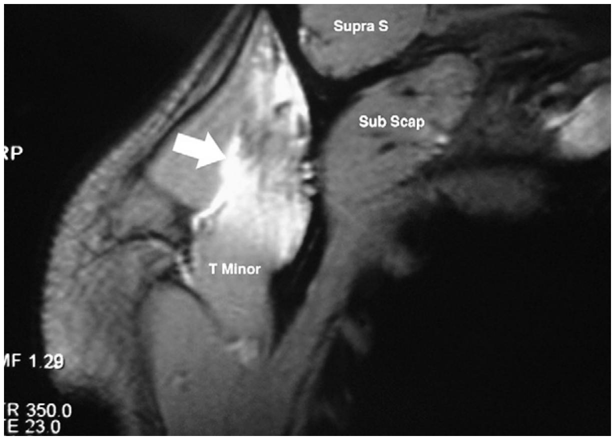

Figure 6 Sagittal fat saturated T1 weighted MRI with intravenous gadolineum contrast showing high signal in the infraspinatus muscle (*white arrow*). (Patient 2, Table I): *T Minor*, Teres Minor; *S Scap*, Subscapularis; *Supra S*, Supraspinatus.

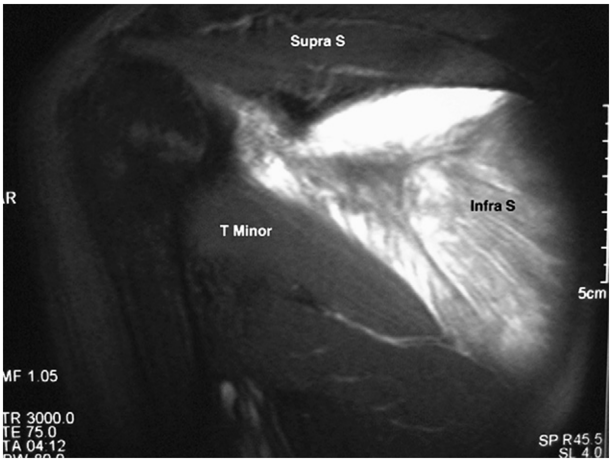

Figure 5 Coronal T2 weighted MRI scan showing edema in the infraspinatus muscle, and a large area of uniform high signal superiorly which may represent a hematoma. (Patient 2, Table I): *T Minor*, Teres Minor; *Supra S*, Supraspinatus.

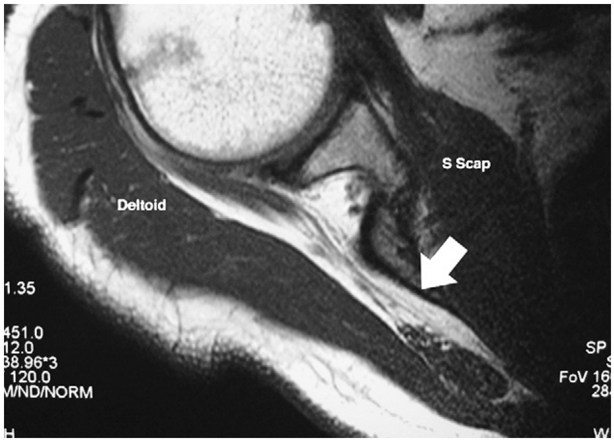

Figure 7 Axial T1 weighted MRI scan showing stage 4 fatty infiltration in the infraspinatus muscle (*white arrow*). (Patient 4, Table I): *S Scap*, Subscapularis.

consisted of supraspinatus tendinitis (4); bursal side partial thickness supraspinatus tears (3); articular side partial thickness supraspinatus tears (3), and 1 musculotendinous tear of supraspinatus. Among the 5 patients, 3 had an intact tendon, while 2 had a full thickness tear of the infraspinatus tendon; however, all 5 developed stage 4 fatty infiltration of the infraspinatus muscle.

DISCUSSION

We describe a lesion of the infraspinatus muscle causing acute pain and weakness of external rotation. This is characterized on MRI by a tendinous or musculotendinous lesion with edema of the infraspinatus muscle progressing to stage four fatty infiltration. In the literature, there have been 2 cases of isolated bony avulsions of the infraspinatus following trauma,[27] and only 1 report of musculotendinous rupture of the supra and infraspinatus muscles, also seen after trauma.[17] These 3 cases describe traumatic entities, which involved only 3 of 19 patients in our study, none of whom had a bony avulsion.

There is no clinical series in the literature of isolated edema of a non-neurological origin occuring in the infraspinatus muscle. In our series, we found no evidence of any previously documented cause of muscular edema. In particular, there were no signs of a neurological etiology on clinical examination or

Figure 8 Sagittal T1 weighted MRI scan showing stage 4 fatty infiltration in the infraspinatus muscle (*white arrow*). (Patient 4, Table I): *S Scap*, Subscapularis; *T Minor*, Teres Minor; *Supra S*, Supraspinatus.

electrophysiological studies. No patient showed any clinical evidence of a systemic myositis, such as dermatomyositis or inclusion body myositis.

Remarkably, there are no reported cases of rotator cuff tears, either tendinous or musculotendinous, associated with edema. It unequivocally supports the finding of edema in tendinous and musculotendinous disruption in other muscle groups.[10,9,30] Tendon tears or avulsions in the rotator cuff are well known to produce fatty infiltration, either in the directly affected muscle or in the adjacent infraspinatus, in the case of supraspinatus tendon tears.[15,24] Architectural changes following alterations in muscle tension and pennation angles have been postulated as the cause of this fatty infiltration.[25,23] The evidence suggesting that grade 3 musculotendinous disruption progresses to fatty infiltration is largely anecdotal. Deutsch et al[9] reported a single case of post-traumatic fatty infiltration after quadriceps trauma, and others, such as Palmer,[28] ascribe this lack of documentation to the oberservation that "in the face of an obvious clinical diagnosis imaging is rarely carried out." It is logical to assume that complete musculotendinous disruption leads to major muscle architectural changes and subsequent fatty infiltration. Unfortunately, there is no major body of published evidence to support this supposition. In our series, the progression in the infraspinatus muscle from edema to fatty infiltration was directly observed in all 19 patients.

The etiology of this condition is not clear. Trauma directly related to rupture occured in only 2 of our patients; these patients appear to be a distinct subgroup. Five patients related their symptom onset to aspiration or injection of their shoulder. All 5 had edema at their next MRI, which was done on an average of 4.5 months (range, 2.9-5.7) after the event. Muscular edema is transitory, rarely persisting over 1 year in both neurological and traumatic injuries.[12] The suggestion that the intervention caused the rupture is not proven. It may have been the rupture that precipitated the pain, leading them to seek acute medical treatment. In 10 of the other 12 cases with progressive onset and chronic shoulder pain, a minor traumatic event occurred and was felt by the patients to be the trigger for the increased severity of their pain. The minor degree of trauma, typically a fall at ground level with the affected arm outstreched, suggests an acute on chronic episode on a backgound of a degenerative etiology. The cause of this degeneration in this young population is unknown. The high incidence of associated cuff pathology, with 90% of our patients having either intratendinous calcification, supraspinatus tendinitis, or partial thickness rotator cuff tears, may be an integral part of this condition. It is known that calcific tendinitis occurs more frequently in females than males, with a peak incidence between 31-40 years of age.[36] The incidence in asymptomatic individuals has been estimated at 2.7%, with this rising to 6.8% in painful shoulders.[2] The high incidence of calcific tendinitis (68%), the younger age of our patient group, and the preponderance of females suggests that it has a role in this condition. As not all patients had calcific deposits, it is not the only cause, but it may intrinsically weaken the tendon or lead to it being weakened by multiple corticoid injections.

Because of the design of our study, we were unable to establish the exact site of the shoulder injections. Corticosteroid injections have an important anti-inflamatory and analgesic effect; however, in an animal model, they have been shown to weaken normal tendon.[34,35] It is also well-known that intratendinous injection may result in rupture.[18,31] The exact role steroid injections play in this condition is unknown, and while we are unable to prove that there is a casual relationship with all cases, they may be a significant factor in some patients. The small numbers who underwent surgery do not allow us to draw firm conclusions. Only 5 patients had surgical repair of the infraspinatus muscle-tendon unit; all 5 had no postoperative improvement in function and all developed severe fatty infiltration. Based on these early findings, we have stopped performing surgical repair for this condition.

The acute on chronic onset of severe pain in the presence of calcific tendinitis would logically reinforce the diagnosis of an acute calcific tendinitis. The rupture of the infraspinatus could be easily overlooked. However, wasting of the infraspinatus muscle and weakness in external rotation, associated with MRI findings of infraspinatus rupture, edema, and fatty infiltration, are almost diagnostic. Its predominantly extra-articular nature means that it can remain undiagnosed, even at the time of arthroscopic exploration, where it is mandatory to explore the posterior part of the cuff on the bursal side. During the same period

of time, we collected 41 other cases of isolated grade 4 fatty infiltration of the infraspinatus with lesions confined to the infraspinatus. None of those patients had any edema because the imaging studies were based on CT arthrograms or because the first MRI was performed after resolution of edema. These patients were not included in this series; however, the final condition of the infraspinatus muscle supports the hypothesis that they are part of the same pathological process and suggests that this condition could be more common than reported and is probably frequently overlooked.

We have identified an isolated, full-thickness lesion of the infraspinatus. Its pathology and natural history appear to be twofold: 1) the rare occurrence of a true traumatic rupture and, more commonly, 2) the patient with chronic shoulder pathology, who has an insult such as a minor fall or iatrogenic event such as an injection or aspiration of a calcific deposit, the final common pathway being disruption of the infraspinatus between the musculotendinous junction and its humeral insertion. The result in all cases is severe initial pain associated with muscular edema on MRI and late fatty infiltaration and wasting of the infraspinatus muscle. Conservative treatment results in chronic weakness of external rotation and some residual discomfort or pain. Surgery on the 5 operative patients was not any better or worse.

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